

(FILE 'HOME' ENTERED AT :53:35 ON 29 OCT 2002)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 12:54:05 ON 29 OCT 2002

L1	10964 S (RETINOBLASTOMA PROTEIN)
L2	576 S L1 AND ANTIBODIES
L3	141 S L2 AND CDK?
L4	77 S L3 AND CDK2
L5	40 S L4 AND CDK4
L6	20 DUPLICATE REMOVE L5 (20 DUPLICATES REMOVED)

L/C  
10/28/02

L6 ANSWER 12 OF 20 MEDLINE  
 AN 1999406807 MEDLINE  
 DN 99406807 PubMed ID: 10477583  
 TI B cell antigen receptor-mediated activation of cyclin-dependent  
**retinoblastoma protein** kinases and inhibition by  
 co-cross-linking with Fc gamma receptors.  
 AU Tanguay D; Pavlovic S; Piatelli M J; Bartek J; Chiles T C  
 CS Department of Biology, Boston College, Chestnut Hill, MA 02467, USA.  
 NC AI-34586 (NIAID)  
 SO JOURNAL OF IMMUNOLOGY, (1999 Sep 15) 163 (6) 3160-8.  
 Journal code: 2985117R. ISSN: 0022-1767.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199910  
 ED Entered STN: 19991014  
 Last Updated on STN: 20020420  
 Entered Medline: 19991004  
 AB Cross-linking the B cell Ag receptor (BCR) to surface Fc receptors for IgG  
 (Fc gamma R) inhibits G1-to-S progression; the mechanism by which this  
 occurs is not completely known. We investigated the regulation of three  
 key cell cycle regulatory components by BCR-Fc gamma R co-cross-linking:  
 G1-cyclins, cyclin-dependent kinases (**Cdks**), and the  
 retinoblastoma gene product (Rb). Rb functions to suppress G1-to-S  
 progression in mammalian cells. Rb undergoes cell-cycle-dependent  
 phosphorylation, leading to its inactivation and thereby promoting S phase  
 entry. We demonstrate in this paper for the first time that BCR-induced Rb  
 phosphorylation is abrogated by co-cross-linking with Fc gamma R. The  
 activation of **Cdk4/6**- and **Cdk2**-dependent Rb protein  
 kinases is concomitantly blocked. Fc gamma R-mediated inhibition of  
**Cdk2** activity results in part from an apparent failure to express  
**Cdk2** protein. By contrast, inhibition of **Cdk4/6**  
 activities is not due to suppression of **Cdk4/6** or cyclins D2/D3  
 expression or inhibition of **Cdk**-activating kinase activity.  
**Cdk4**- and **Cdk6**-immune complexes recovered from B cells  
 following BCR-Fc gamma R co-cross-linking are devoid of coprecipitated  
 D-type cyclins, indicating that inhibition of their Rb protein kinase  
 activities is due in part to the absence of bound D-type cyclin. Thus,  
 BCR-derived activation signals that up-regulate D-type cyclin and  
**Cdk4/6** protein expression remain intact; however, Fc gamma  
 R-mediated signals block cyclin D-**Cdk4/6** assembly or  
 stabilization. These results suggest that assembly or stabilization of  
 D-type cyclin holoenzyme complexes 1) is an important step in the  
 activation of **Cdk4/6** by BCR signals, and 2) suffice in providing  
 a mechanism to account for inhibition of BCR-stimulated Rb protein  
 phosphorylation by Fc gamma R.  
 CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't,  
 Non-P.H.S.; Support, U.S. Gov't, P.H.S.  
**Antibodies, Anti-Idiotypic: PD, pharmacology**  
 B-Lymphocytes: EN, enzymology  
 B-Lymphocytes: IM, immunology  
 B-Lymphocytes: ME, metabolism  
 Cell Differentiation: IM, immunology  
 Cyclin E: AI, antagonists & inhibitors  
 Cyclin E: BI, biosynthesis  
 \*Cyclin-Dependent Kinases: AI, antagonists & inhibitors  
 Cyclin-Dependent Kinases: BI, biosynthesis  
 \*Cyclin-Dependent Kinases: ME, metabolism  
 Cyclins: AI, antagonists & inhibitors  
 Cyclins: BI, biosynthesis  
 DNA: AI, antagonists & inhibitors  
 DNA: BI, biosynthesis  
 Enzyme Activation: IM, immunology  
 G1 Phase: IM, immunology  
 Holoenzymes: BI, biosynthesis  
 Immunoglobulins, Fab: PD, pharmacology  
 Mice  
 Mice, Inbred BALB C  
 Microtubule-Associated Proteins: BI, biosynthesis  
 Phosphorylation  
 Protein-Serine-Threonine Kinases: AI, antagonists & inhibitors

• **Protein**); 0 (cyclin D); EC 2.7.1.- (CAK1 protein); EC 2.7.1.- (  
**CDK2** protein); EC 2.7.1.- (**CDK6** protein); EC 2.7.1.-  
(Protein-Serine-Threonine Kinases); EC 2.7.1.- (p34PSK- kinase); EC  
2.7.1

6 ANSWER 17 OF 20 CAPLU COPYRIGHT 2002 ACS DUPLICATE 7  
AN 1997:75422 CAPLUS  
DN 126:155793  
TI Monoclonal **antibodies** specific for underphosphorylated  
**retinoblastoma protein** identify a cell cycle regulated  
phosphorylation site targeted by **CDKs**  
AU Zarkowska, Tamara; U, Sally; Harlow, Ed; Mittnacht, Sibylle  
CS Department of Cell and Molecular Biology, Institute of Cancer Research,  
London, SW3 6JB, UK  
SO Oncogene (1997), 14(2), 249-254  
CODEN: ONCNES; ISSN: 0950-9232  
PB Stockton  
DT Journal  
LA English  
CC 14-1 (Mammalian Pathological Biochemistry)  
Section cross-reference(s): 15  
AB The growth suppressive activity of the retinoblastoma tumor suppressor  
protein is controlled by cell cycle dependent phosphorylation. However,  
while many in vivo phosphorylation sites have been mapped, the identities  
of those residues whose phosphorylation is regulated remain elusive. We  
have mapped the epitopes of three independent monoclonal  
**antibodies** that recognize a distinction between differentially  
phosphorylated pRB sub-populations. All three **antibodies**  
recognize an identical epitope which encompasses an essential serine  
positioned within a consensus site for proline directed kinase  
phosphorylation. We provide evidence that this residue, serine 608 of  
pRB, is an authentic phosphorylation site that can be phosphorylated in  
vitro by cyclin A-**CDK2** and cyclin D1-**CDK4** kinases but  
not by cyclin E-**CDK2** kinase or the mitogen activated kinase  
ERK2. Phosphorylation at this residue seems to be cell cycle regulated,  
occurring prior to entry into the S phase.  
ST phosphorylation site **CDK** kinase **retinoblastoma**  
**protein**; monoclonal antibody phosphorylation  
**retinoblastoma protein**  
IT Cyclins  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(A; monoclonal **antibodies** specific for underphosphorylated  
**retinoblastoma protein** identify a cell cycle  
regulated phosphorylation site targeted by **CDKs**)  
IT Cyclins  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(D1; monoclonal **antibodies** specific for underphosphorylated  
**retinoblastoma protein** identify a cell cycle  
regulated phosphorylation site targeted by **CDKs**)  
IT Transcription factors  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(Rb; monoclonal **antibodies** specific for underphosphorylated  
**retinoblastoma protein** identify a cell cycle  
regulated phosphorylation site targeted by **CDKs**)  
IT Cell cycle  
(monoclonal **antibodies** specific for underphosphorylated  
**retinoblastoma protein** identify a cell cycle  
regulated phosphorylation site targeted by **CDKs**)  
IT Protein motifs  
(phosphorylation site; monoclonal **antibodies** specific for  
underphosphorylated **retinoblastoma protein** identify  
a cell cycle regulated phosphorylation site targeted by **CDKs**)  
IT 141349-86-2, **Cdk2** kinase 147014-97-9, **Cdk4** kinase  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(monoclonal **antibodies** specific for underphosphorylated  
**retinoblastoma protein** identify a cell cycle  
regulated phosphorylation site targeted by **CDKs**)  
IT 56-45-1, Serine, properties  
RL: PRP (Properties)  
(monoclonal **antibodies** specific for underphosphorylated  
**retinoblastoma protein** identify a cell cycle  
regulated phosphorylation site targeted by **CDKs**)

12 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2002 ACS  
 AN 2001:115399 CAPLUS  
 DN 134:174844  
 TI Method for assaying phosphorylation enzymatic activity of cyclin/CDK complex  
 IN Suzuki, Susumu; Tamai, Katsuyuki; Toji, Shingo; Ogawa, Akira  
 PA Medical & Biological Laboratories Co., Ltd., Japan  
 SO PCT Int. Appl., 63 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA Japanese  
 IC ICM G01N033-573  
 ICS G01N033-50; G01N033-15; C12Q001-48; C07K007-08; C07K016-18  
 CC 7-1 (Enzymes)  
 Section cross-reference(s): 1, 15

*applicant*

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001011367	A1	20010215	WO 2000-JP5219	20000803
	W: JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	EP 1207395	A1	20020522	EP 2000-949981	20000803
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
PRAI	JP 1999-221612	A	19990804		
	WO 2000-JP5219	W	20000803		
AB	A method is provided for assaying a retinoblastoma (RB) protein-phosphorylating enzymic activity of a cyclin/CDK (cyclin-dependent kinase) complex (e.g., cyclinA/CDK1, cyclinA/CDK2, cyclinB/CDK1, cyclinD1/CDK4, cyclinD1/CDK6, cyclinD2/CDK4, cyclinD2/CDK6, cyclinD3/CDK4, cyclinD3/CDK6, cyclinE/CDK2) by evaluating the phosphorylation of RB protein by an immunol. method (e.g., <b>ELISA</b> ) using <b>antibodies</b> capable of recognizing the phosphorylated state of RB protein. A method is also provided for assaying a dephosphorylating enzymic activity of the cyclin/CDK complex toward the RB protein phosphorylated by the cyclin/CDK complex. Antigens for producing the <b>antibodies</b> used for these methods are also claimed. These antigens contain the peptide consisting of the amino acid sequence contg. the phosphorylation site (356th threonine, 612nd serine, 780th serine, 807th threonine) of the RB protein in a phosphorylated state. These methods are applied to screening a compd. capable of adjusting these enzymic activities.				
ST	<b>retinoblastoma protein</b> phosphorylation cyclin CDK complex; cyclin dependent kinase immunoassay RB protein				
IT	Cyclins				
	RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process) (A; B; D1; D2; D3; E; method for assaying phosphorylation enzymic activity of cyclin/CDK complex)				
IT	Transcription factors				
	RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process) (Rb; method for assaying phosphorylation enzymic activity of cyclin/CDK complex)				
IT	Immunoassay				
	(enzyme-linked immunosorbent assay; method for assaying phosphorylation enzymic activity of cyclin/CDK complex)				
IT	Dephosphorylation, biological				
	Drug screening				
	Immobilization, biochemical				
	Immunoassay				
	Test kits				
	(method for assaying phosphorylation enzymic activity of cyclin/CDK complex)				
IT	Peptides, biological studies				
	RL: BUU (Biological use, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); USES (Uses) (method for assaying phosphorylation enzymic activity of cyclin/CDK complex)				
	Primers (nucleic acid)				
	RL: NUU (Other use, unclassified); USES (Uses)				

IT Phosphorylation, biological  
(protein; method for assaying phosphorylation enzymic activity of cyclin/CDK complex)

IT 150428-23-2D, Cyclin-dependent kinase, complex with cyclinD1; complex with cyclinD2; complex with cyclinD3  
RL: ANT (Analyte); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
(6; method for assaying phosphorylation enzymic activity of cyclin/CDK complex)

IT 325789-94-4P 325789-95-5P 325789-96-6P 325789-97-7P  
RL: BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)  
(amino acid sequence; method for assaying phosphorylation enzymic activity of cyclin/CDK complex)

IT 143375-65-9D, Cyclin-dependent kinase 1, complex with cyclinA; complex with cyclinB  
RL: ANT (Analyte); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)  
(method for assaying phosphorylation enzymic activity of cyclin/CDK complex)

IT 141349-86-2D, Cyclin-dependent kinase 2, complex with cyclinA; complex with cyclinE 147014-97-9D, Cyclin-dependent kinase 4, complex with cyclinD1; complex with cyclinD2; complex with cyclinD3  
RL: ANT (Analyte); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
(method for assaying phosphorylation enzymic activity of cyclin/CDK complex)

IT 56-45-1, Serine, biological studies 72-19-5, Threonine, biological studies  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(method for assaying phosphorylation enzymic activity of cyclin/CDK complex)

IT 325868-62-0, 1: PN: WO0111367 SEQID: 9 unclaimed DNA 325868-63-1, 2: PN: WO0111367 SEQID: 10 unclaimed DNA 325868-64-2, 3: PN: WO0111367 SEQID: 11 unclaimed DNA 325868-65-3, 4: PN: WO0111367 SEQID: 12 unclaimed DNA 325868-66-4, 5: PN: WO0111367 SEQID: 13 unclaimed DNA 325868-67-5, 6: PN: WO0111367 SEQID: 14 unclaimed DNA 325868-68-6, 7: PN: WO0111367 SEQID: 15 unclaimed DNA 325868-69-7, 8: PN: WO0111367 SEQID: 16 unclaimed DNA 325868-70-0, 9: PN: WO0111367 SEQID: 17 unclaimed DNA 325868-71-1 325868-72-2 325868-73-3  
RL: PRP (Properties)  
(unclaimed nucleotide sequence; method for assaying phosphorylation enzymic activity of cyclin/CDK complex)

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Seiji, O; Ketsueki Shuyouka 1996, V32(2), P123

(2) Shinichi, I; Nishinippon Hifuka 1995, V57(4), P687

12 ANSWER 3 OF 7 MEDLINE DUPLICATE 2  
AN 1999402221 MEDLINE  
DN 99402221 PubMed ID: 10475236  
TI Immunohistochemical analysis of the D-type cyclin-dependent kinases Cdk4 and Cdk6, using a series of monoclonal **antibodies**.  
AU Lukas C; Jensen S K; Bartkova J; Lukas J; Bartek J  
CS Institute of Cancer Biology, Danish Cancer Society, Copenhagen.  
SO HYBRIDOMA, (1999 Jun) 18 (3) 225-34.  
Journal code: 8202424. ISSN: 0272-457X.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199910  
ED Entered STN: 19991101  
Last Updated on STN: 19991101  
Entered Medline: 19991021  
AB Cellular signal transduction cascades triggered by mitogenic or antiproliferative cues eventually converge on a biochemical mechanism centered around the retinoblastoma tumor suppressor (pRb), the so-called RB pathway that governs G1-phase progression and guards the commitment to enter S phase. pRb, together with its immediate upstream regulators, the D-type cyclins, their partner cyclin-dependent kinases Cdk4 and Cdk6, and the Cdk inhibitors, form a functional unit that is involved in major decisions about cellular fate, and whose components, including the proto-oncogenic cyclin D-dependent kinases, are commonly deregulated in many types of cancer. We report here the production and characterization of a series of 12 monoclonal **antibodies** (MAbs) that specifically recognize either Cdk4 or Cdk6. These **antibodies** are proving to be invaluable molecular probes for defining abundance, subcellular localization, binding partners, and ultimately the function(s) of these cell cycle-regulatory kinases. Localization of the target epitopes was mapped by peptide enzyme-linked immunoadsorbent assay (**ELISA**), and two **antibodies** recognizing sequences adjacent to N-terminus of Cdk4 can discriminate between the wild-type protein and the oncogenic, melanoma-associated R24C mutant of this kinase. Individual **antibodies** of our panel recognize distinct pools of Cdk4/6, a feature reflected by their differential applicability in immunoblotting, immunoprecipitation, kinase assays, and immunostaining including immunohistochemistry on archival paraffin-embedded tissue sections. Collectively, the **antibodies** described in this study provide the means for immunochemical analyses of the cyclin D-dependent kinases in human and animal cells, and represent useful molecular tools that should help better understand the biological roles of Cdk4 and Cdk6 in normal cell-cycle control, and their oncogenic activity in tumor cells.  
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't  
**\*Antibodies, Monoclonal**  
Base Sequence  
Cell Line  
\*Cyclin-Dependent Kinases: CH, chemistry  
Cyclin-Dependent Kinases: GE, genetics  
\*Cyclin-Dependent Kinases: IM, immunology  
DNA Primers: GE, genetics  
Epitope Mapping  
Hybridomas: IM, immunology  
Immunochemistry  
Immunohistochemistry  
Mice  
Mutation  
Neoplasms: EN, enzymology  
Neoplasms: GE, genetics  
\*Protein-Serine-Threonine Kinases: CH, chemistry  
\*Protein-Serine-Threonine Kinases: IM, immunology  
Rats  
**Retinoblastoma Protein: ME, metabolism**  
CN 0 (**Antibodies, Monoclonal**); 0 (Cyclin-Dependent Kinases); 0 (DNA Primers); 0 (**Retinoblastoma Protein**); EC 2.7.1.- (CDK6 protein); EC 2.7.1.- (Protein-Serine-Threonine Kinases); EC 2.7.1.- (p34P

L12 ANSWER 7 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3  
 AN 1994:226130 BIOSIS  
 DN PREV199497239130  
 TI **Retinoblastoma protein** monoclonal **antibodies**  
 with novel characteristics.  
 AU Wen, Shu Fen; Nodelman, Margarita; Nared-Hood, Karen; Duncan, John;  
 Geradts, Joseph; Shepard, H. Michael (1)  
 CS (1) Dep. Assay Dev., Canji Inc., 3030 Science Park Road, Suite 302, San  
 Diego, CA 92121 USA  
 SO Journal of Immunological Methods, (1994) Vol. 169, No. 2, pp. 231-240.  
 ISSN: 0022-1759.  
 DT Article  
 LA English  
 AB We have developed a family of monoclonal **antibodies** directed  
 against the retinoblastoma gene product (p110-RB). One of these monoclonal  
**antibodies**, 3C8, binds p110-RB near the C-terminal end of the  
 protein (aa886-aa905). It was characterized by immunoblotting,  
**ELISA**, fluorescence-activated flow cytometry and  
 immunohistostaining. It was shown to be useful for the detection of  
 P110-RB in formalin-fixed and paraffin-embedded tissue sections. Because  
 3C8 binds outside of regions shown to be involved in p110-RB interactions  
 with other cellular proteins, it may be an especially useful reagent for  
 the reliable detection of p110-RB in tumor cells, and for the isolation by  
 affinity chromatography of p110-RB complexes with other cellular proteins.  
 CC Microscopy Techniques - Cytology and Cytochemistry 01054  
 Microscopy Techniques - Histology and Histochemistry 01056  
 Cytology and Cytochemistry - Human \*02508  
 Genetics and Cytogenetics - Human \*03508  
 Radiation - Radiation and Isotope Techniques 06504  
 Biochemical Methods - Nucleic Acids, Purines and Pyrimidines \*10052  
 Biochemical Methods - Proteins, Peptides and Amino Acids \*10054  
 Biochemical Methods - Carbohydrates \*10058  
 Biochemical Studies - Nucleic Acids, Purines and Pyrimidines \*10062  
 Biochemical Studies - Proteins, Peptides and Amino Acids 10064  
 Biochemical Studies - Carbohydrates 10068  
 Biophysics - General Biophysical Techniques 10504  
 Biophysics - Molecular Properties and Macromolecules \*10506  
 Enzymes - Methods 10804  
 Pathology, General and Miscellaneous - Diagnostic 12504  
 Nervous System - Anatomy \*20502  
 Nervous System - Pathology \*20506  
 Neoplasms and Neoplastic Agents - Pathology; Clinical Aspects; Systemic  
 Effects \*24004  
 Neoplasms and Neoplastic Agents - Carcinogens and Carcinogenesis \*24007  
 Immunology and Immunochemistry - General; Methods \*34502  
 BC Hominidae \*86215  
 IT Major Concepts  
 Biochemistry and Molecular Biophysics; Cell Biology; Genetics; Immune  
 System (Chemical Coordination and Homeostasis); Methods and Techniques;  
 Nervous System (Neural Coordination); Neurology (Human Medicine,  
 Medical Sciences); Oncology (Human Medicine, Medical Sciences)  
 IT Miscellaneous Descriptors  
 ANALYTICAL METHOD; DIAGNOSIS; **ELISA**; FLUORESCENCE-ACTIVATED  
 CELL SORTER; IMMUNOHISTOCHEMISTRY; MONOCLONAL **ANTIBODY** 3C8;  
 PROGNOSIS; RETINOBLASTOMA GENE PRODUCT; TUMOR SUPPRESSOR GENE; WESTERN  
 BLOT  
 ORGN Super Taxa  
 Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia  
 ORGN Organism Name  
 Hominidae (Hominidae)  
 ORGN Organism Superterms  
 animals; chordates; humans; mammals; primates; vertebrates

L2 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2002 ACS  
 AN 1995:798794 CAPLUS  
 DN 123:191683  
 TI The corral hypothesis: A novel regulatory mode for **retinoblastoma protein** function  
 AU Lee, W. -H.; Xu, Y.; Hong, F.; Durfee, T.; Mancini, M. A.; Ueng, Y. -C.; Chen, P. -L.; Riley, D.  
 CS Institute Biotechnology, University Texas, San Antonio, TX, 78245, USA  
 SO Cold Spring Harbor Symposia on Quantitative Biology (1994), 59(Molecular Genetics of Cancer), 97-107  
 CODEN: CSHSAZ; ISSN: 0091-7451  
 PB Cold Spring Harbor Laboratory Press  
 DT Journal  
 LA English  
 CC 6-3 (General Biochemistry)  
 Section cross-reference(s): 3, 14  
 AB Several pieces of evidence are described to support and strengthen the corral hypothesis. First, the authors demonstrate the ability of the Rb protein to bind specifically, but with different affinities to a group of proteins identified by the yeast two-hybrid system and the "Rb-sandwich" method, as described previously (Shan et al., 1992; Durfee et al., 1993). These proteins may function either during the G1/S transition or during M-phase progression. Second, the authors show that the oligomerized form of Rb protein binds to an assocd. protein and that phosphorylation of Rb protein by Cdks leads to a significant attenuation of the oligomerizing property, suggesting that phosphorylation together with those described previously, provide convincing evidence that Rb protein regulates other nuclear proteins in a unique, specific, and coordinated manner. Subcompartments created by the complexes of Rb protein with other nuclear proteins are postulated to account for phenomena obsd. in human cells and animals.  
 ST **retinoblastoma protein** function corral hypothesis  
 IT Ribonucleic acid formation factors  
 RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 (gene Rb, novel regulatory mode for **retinoblastoma protein** function as described by the corral hypothesis)  
 IT Eye, neoplasm  
 (retinoblastoma, novel regulatory mode for **retinoblastoma protein** function as described by the corral hypothesis)